Trans. Nat. Acad. Sci. & Tech. (Phils.)
1987.9:523-5-39

HEALTH HAZARDS IN THE PLASTIC INDUSTRY PROBLEMS IN RISK ASSESSMENT

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ABSTRACT

The work environment and the health of workers in the Plastic Industry in the Philippines were studied in the last two years using a multiple-step stratified sampling method.

The study indicated that only 5 out of a total of 312 companies and 52 respondents registered in the Association of Plastic Industry companies use the toxic plastic Monomers Vinyl Chloride (VCM) or Styrene (SM) as raw materials or intermediates in their production process. All the others either use polymers from these local companies or import intermediate raw materials in the form of plastic polymer powders and pellets, and fabricate them into various finished products.

The findings show that the work environment in the plastic industry in the Philippines is inadequate with respect to general physical conditions and occupational health and safety measures. These inadequacies notwithstanding, there was no evidence of the Vinyl Chloride Diseases Syndrome characterized by signs and symptoms of peripheral vascular disease particularly in the hands, or increased incidence of pulmonary fibrosis from chronic exposure in dust, in exposed workers.

The evaluation of the finding of an apparent increase in some companies of liver disorders, including hepatocarcinoma will be discussed with respect to the problems of multifactorial risk assessment in the Philippine setting.

Introduction

Since the 1960's as a sector, worldwide, the Plastic Industry has been growing faster than most other sectors. From a world production of 24 million tons in 1969, it grew to 58 million in 1980, and is forecast to reach 100 million tons in 1990 (Encyclopedia of Occupational Health and Safety, 1983).

This extremely rapid growth was brought about by the great usefulness of a variety plastic products in industry and at home, and the availability in great abundance of cheap base chemicals from oil and natural gas. Through the process of cracking and distillation the petrochemical industry has provided monomeric raw materials which when polymerized are transformed to the familiar thermoplastics like polyvinylchloride or polyethylene and the less familiar thermosetting resins used in paints and glues.

Starting in the 1960's small, medium and large plastic companies were established in the country which numbered more than 300 registered companies in 1981.

But along with the benefits obtained from the multitude of plastic products, come the cost and the hazards. Some time in the 1970's a clinical syndrome characterized by clubbing of the fingers and acro-osteolysis demonstrable by X-ray, was associated with chronic exposure to Vinyl Chloride Monomer (VCM). This Syndrome came to be known as the Vinyl Chloride Disease Syndrome. (N.Y. Acad. of Sci., 1975). More ominous than this was the association of chronic high level VCM exposure with the development of liver disorders including primary liver cancer particularly a specific type-Angiosarcoma of the Liver (Creech and Johnson, 1974).

With the continuous growth of the plastic industry in the Philippines, it became important to know the status of the work environment and the health of workers in the industry. Thus, this study funded by the Philippine Council for Health Research and Development, was undertaken starting in 1984.

Materials and Methods

A modified multi-step stratified sampling method was used in the selection of companies and subjects included in the sample studied.

Three hundred thirteen (313) registered Plastic companies were surveyed of which only 52 responded positively. From this 52 responders, 23 companies representing small, medium, and large companies located in Luzon, Visayas and Mindanao were selected. Of the 23, 10 companies engaged in the production or use of VCM, polyvinyl chloride (PVC) styrene or polystyrene, were included for in-depth studies of the work environment and the health of workers.

The studies/measurements/activities done under this project were the following:

a) Survey of the plastic industry with special attention to the work environment and the health of workers. n = 52

b) Plant visits to selected companies representing a cross section of the plastic industry. Majority of these companies are located in Metro Manila, others in Cebu, Davao and Iligan. n = 23

c) Review of medical records on Accidents, Absenteeism Morbidity and Mortality from 1980-1983. n = 10

d) Measurement of the concentration in the air of vinyl chloride and styrene in the production and control areas of plastic companies. n = 10

e) Physical medical examination of selected subjects in control and experimental groups. n_t = 241

Laboratory examination of control and experimental groups including:

n_t = 241

1. Blood Chemistry
   - SGPT
   - BUN
   - LDH

2. Blood Counts
   - RBC and Hct.
   - WBC and differential
   - Platelets

g) X-rays of Control and Experimental Groups. $n_t = 241$
   1. Chest
   2. Hands

h) Retrospective Review of the National Central Tumor Registry
i) Education and Information Campaign (Posters)

Results and Findings

Profile of plastic companies

The results of the survey show that most of the companies are engaged in processing and fabrication (18) and only (5) are in polymer or resin manufacturing either solely (1) or in combination with processing and fabrication (4). Table 1.

Table 1. Classification of plastic companies by types of production

Type of production	*Number*	*%*
A. Polymer/Resin Manufacturing	1	4.4
B. Processing and Fabrication	18	78.3
C. Combination of A & B	4	17.4
Total	23	100

The range of raw materials used in the 23 companies included in this study are listed in Table 2.

Three or 13% used VCM, 5 or 22% used PVC and the majority 13 or 57% used a combination of two or more raw materials.

Table 2. Distribution of plastic companies surveyed according to raw materials used

Raw materials	*No. of plastic companies*	*%*
Vinyl Chloride Monomer	3	13
Poly Vinyl Chloride	5	22
Styrene	2	8
Combinations:		
Styrene Polystyrene Polyethylene Etc.	13	57

Work environment and health facilities

Qualitative assessment of the physical condition of the work in the 23 companies visited by the project team showed that 83% has satisfactory lighting, 74% has satisfactory cleanliness and housekeeping.

Table 3. State of the work environment in plastic companies visited

Parameter	n	*Assessment*			*%*
		Satisfactory	*%*	*Unsatisfactory*	
A. Ventilation	23	9	39	14	61
B. Lighting	23	19	83	4	17
C. Cleanliness and Housekeeping	23	17	74	6	26

As far as facilities, protective measures and health services for the employees are concerned, 86% had satisfactory medical staff mostly on retainer arrangement on part-time basis, 59% maintained a medical clinic, and 45% kept some medical records. However, from all indications, the occupational health program and the quality of medical services offered still left much to be desired even in those companies where facilities were available.

Toilet and canteen facilities were also largely inadequate in most companies except for the largest ones.

Protective devices were provided in 55% of the companies visited but were not of high quality.

Record of accidents, morbidity and mortality

The accident rate in 1980 ranged from 25 to 42 per 1000 persons at risks. The morbidity records among the workers on the plastic industry showed the

normal pattern of illnesses in the general population (Table 4). According to Reverente (1982) only 3-5% of all sick leaves in local companies including industries, other than plastic are directly work-related. Ninety five percent (95%) of absences from work are due to non-occupational illnesses such as viral respiratory infection, etc.

Table 4. Morbidity cases of 23 plastic companies visited for the period 1981-1983

Health Problem	*1981*	*1982*	*1983*	*Total*
Respiratory	2,869	4,149	1,544	8,562
Gastrointestinal	1,111	1,341	304	2,756
Dermatological	376	173	198	717
Hypersensitivity	177	186	45	408
Ophthalmological	248	94	31	373
Cardiovascular	108	219	84	411
Gynecological	98	126	58	282
Skeletal	56	79	19	154
Central Nervous System	48	52	42	142
Hematological	11	26	8	45

Concentration of vinyl chloride and styrene monomer in air samples from various locations in the work environment

The qualitative measurement of Vinyl Chloride and Styrene monomer in production and control areas using the Kitagawa Detector tubes and Sampling Pump showed that Threshold Limit Value (1 ppm) were frequently exceeded in some companies engaged in the polymer manufacturing using vinyl chloride monomer as raw materials (Table 5). In one company engaged in both the synthesis of vinyl chloride monomer and production of polyvinyl chloride the short time exposure limit or ceiling concentration was also exceeded in the automated control room for the production process.

From a report of the U.S. Environmental Protection Agency the typical air concentration in various locations in a Vinyl Chloride/Chloride Polyvinyl Chloride plant are shown in Table 6.

Signs and symptoms of acute poisoning

The progressive signs and symptoms associated with various concentrations of Vinyl Chloride Monomer is shown in Table 7. The odor threshold is already at the fairly high concentration of 2,000 ppm. Behavioral effects and subjective sensations of elation, asthenia and heaviness of the legs are seen with concentration in the range of 5,000 to 8,000 ppm and possible death at 120,000 ppm. In our investigation, there was one reported case of a worker falling accidentally into the reactor vessel while cleaning it. Whether this was due to acute intoxication remains to be proven.

Table 5. Concentration of vinyl chloride and styrene monomers in various areas of ten (10) companies under study

Area	*Gas Sample Concentration*										
	Vinyl Chloride					*Styrene*				*Acrylo-nitrile*	
	PPM					*PPM*				*PPM*	
Extruder	5	.05	–	–	–*	–	–	3	5	5	neg.
Mixing	1	–	–	3	–	–	3	–	–	–	–
Molding	–	–	–	–	–	–	–	4	10	2	4
Reactor	–	–	neg.	–	–	–	–	–	–	–	–
Engineering	–	–	–	neg.	–	–	–	–	–	–	–
Polymerizer	–	–	–	1.5	–	–	–	–	–	–	–
Gas Holder	–	–	1.8	–	–	–	–	–	–	–	–
Kettle	–	–	4	–	–	20	–	–	–	–	–
Blend Tank	–	–	–	–	3	–	–	–	–	–	–

Table 6. Typical concentration of vinyl chloride**

	mg/m^3	*ppm*
Reactor prior to ventilation	7,800 mg/m^3	3000
Reactor during scraping	130-260	50-100
Near hands during scraping	1,560-2,600	600-1000

* Monitoring results were taken by the company dated April 18 and May 1, 1984.
**Reference: U.S. Environmental Protection Agency, 1974.

Table 7. Signs and symptoms of acute poisoning with vinyl chloride monomer*

Concentration	*S & S*
100 ppm	Not Perceptible
2,000 – 5,000 ppm	Odor Threshold = Sweetish Odor
5,000 ppm	Elation Asthenia Heaviness in Legs and Somnolence
8,000 – 10,000 ppm	Vertigo
16,000 ppm	Impaired Hearing and Vision
70,000 ppm	Narcosis
120,000 ppm	May be Fatal

*Reference: New York Academy of Science: 246; 1975, pp. 1-322.

Physical and medical examination

The physical and medical examination of both 97 control in non-production areas and 144 experimental subjects in production areas showed essentially normal and findings without any evidence of hepatosplenomegaly of peripheral vascular disease characteristic of vinyl chloride disease as described in the literature.

Laboratory examination

Blood chemistry namely SGPT, BUN, and LDH, and blood counts, namely RBC and hematocrit, WBC and Differential and Platelet counts, showed results in control and experimental groups which were mostly within normal range and not significantly different from each other (Table 8-SGPT, BUN, LDH and Table 9-RBC, hemacrit, WBC, Differential Platelets).

Table 8. Mean values with standard deviation of mean for blood chemistries of both exposed and unexposed groups

Blood Chemistry	*Number*	*Unexposed Group*	*Number*	*Exposed Group*
BUN	97	4.0582 ± .113	144	4.0801 ± .110
ALAKALINE PHOSPHATASE	97	31.106 ± 1.228	144	32.662 ± 1.031
LDH	27*	99.734 ± 7.303	41*	108.141 ± 5.816
SGPT	97	17.325 ± 2.1084	144	19.866 ± 1.844

Table 9. Mean values with standard deviation of mean for blood count for both exposed and unexposed groups

Blood Test	*Number*	*Unexposed Group*	*Number*	*Exposed Group*
Hemoglobin	97	12-586 ± .243	144	13.513 ± .1834
Hematocrit	97	43.248 ± .626	144	42.986 ± .511
WBC	97	8,001.389 ± 344.896	144	8,005.4622 ± 221.14
Lymphocytes	97	39.528 ± .9212	144	40.555 ± .8566
Segmenters	97	58.083 ± .925	144	56.244 ± .8096
Monocytes	97	.375 ± .0801	144	.4538 ± .068
Eosinophil	97	2.125 ± .2712	144	2.7981 ± .3399
Platelet	97	265.750 ± 11.701924	144	254.773.109 ± 9.224466

*Only 27/97 of unexposed group and 41/144 of exposed group were tested for LDH due to unavailability of reagents.

X-rays of the lungs and hands

The X-rays of the lungs and hands of subjects belonging to the control (not at risk) and experimental (at risk) groups showed no tell-tale findings suggestive at lung fibrosis or acroosteolysis which is characteristic of vinyl chloride disease syndrome.

Retrospective review of liver cancer

As a side study, a review of the National Tumor Registry of the Philippine Cancer Society from 1980-1983 was undertaken, which showed 313 cases of liver cancer including 24 cases of hepatocellular and one case of angiosarcoma of the liver (Table 10). Of the total, 97 cases of various types of hepatic cancer were investigated further by going back to their medical charts in 8 hospitals in Metro Manila. In no case was there a definite occupational history suggestive of plastic chemical induced cancer.

Table 10. Hepatic cancer cases taken from the National Central Tumor Registry of the Philippine Cancer Society for the period 1980-1983

Kinds of Liver Cancer	*Year*				
	1980	*1981*	*1982*	*1983*	*Total*
Hepatoma	31	38	52	5	126
Hepatocarcinoma	13	21	26	1	61
Carcinoma	13	10	12	2	37
Hepatocellular CA	3	6	14	1	24
Adenocarcinoma	2	6	8	2	18
Liver Cell Carcinoma	8	2	3	2	15
Malignant Cells	4	–	2		6
Sq. Cell Carcinoma	–	–	4	1	5
Fibrosarcoma	1	–	2		3
Liver Cancer		–	3	2	5
Hepatic Carcinoma	1	–	1	1	3
Carcinoma Sm. Cell	1	–	–	–	1
Metastatic Anaplastic CA	1	–	–	–	1
Metastatic Cell CA	–	1	–	–	1
Analplasticism Cell CA	–	1	–	–	1
Hepatic Cell Carcinoma	–	1	–	–	1
Islet Cell Tumor	–	1	–	–	1
Hepathocholangid CA	–	–	1	–	1
Epidermal Cancer	–	–	1	–	1
Angiosarcoma	–	–	–	1	1
Total	77	87	131	18	313

However, further investigation in one hospital in Cebu City showed that there were two cases of hepatoma and a single case of pathologically proven angiosarcoma of the liver, which was possibly work-related.

The first case of occupational liver angiosarcoma was discovered in the USA in 1961 in a plastic industry worker with 15 years exposure but the work-relatedness of angiosarcoma of the liver to plastic chemical exposure was not substantiated until 1974 by the work of Creech and Johnson.

Subsequently, cases from other parts of the world have been reported totalling now about 90 cases of occupational angiosarcoma of the liver.

Summary and Conclusion

a) Occupational health and safety is not given sufficient attention and priority in the plastic industry as evidenced by the less than adequate health services offered and the fair to poor conditions of the work environment in most of the establishments, as well as evidence that threshold limit value for vinyl chloride monomer in air has been exceeded in some instances.

b) Despite this situation however, the health of the workers at risk compared with a control group was not significantly worse based on the accident rate, morbidity and mortality records from 1981 to 1983, and the results of blood chemistry, blood counts, physical and medical examination, and x-ray of the lungs and hands. These findings suggest that there is still time to take preventive and ameliorative measures to avoid the known hazards of hazardous and toxic chemicals in the plastic industry, particularly vinyl chloride monomer.

c) There was no evidence of vinyl chloride disease such as sclerotic syndrome, acroosteolysis, thrombocytopenia, hepatosplenomegaly and abnormal hepatic function tests among workers in the production areas of plastic companies.

d) From a restrospective side study involving a review of the National Central Tumor Registry and a search made of the medical records of various hospitals in the country, a single case was found of possible occupational angiosarcoma of the liver.

e) In some of the companies especially those using VCM there was evidence of higher incidence of hepatitis and liver cancer than would be expected from the general population.

The problems of primary liver cancer risk assessment

One of the more interesting findings of this study has to do with the apparent increase in liver problems among workers in some plastic companies.

In Company X using VCM as raw material, the Company physician and nurse related to us that at one time in the past, there was a high incidence of liver disorders among workers as. evidenced by elevated blood SGOT enzyme levels. But when we asked for the records of these cases they could not produce them.

In another Company Y using styrene monomer as raw material, located nearby and down wind to Company X, the workers related to us that there was also a time when a large number of workers had complaints related to the liver, and they attributed this to air pollution coming from Company X.

In both cases, there was no record on which to base any conclusion and so these reports remain anecdotal. When we examined the blood enzyme levels of a

sample from the exposed and control groups of these two companies, the levels of SGOT and SGPT were within normal limits and not significantly different between experimental and control groups. Perhaps, had there been a good monitoring and recording system at the time there was the alleged high incidence of liver complaints among workers in these two companies, an epidemic of work-related liver disorders or infectious hepatitis would have been documented.

The problem of risk assessment becomes even more difficult when one deals with a complex condition such as primary liver cancer or hepatocellular carcinoma which is known to have several etiologic factors.

To illustrate this difficulty in risk assessment, take the case of a third large plastic Company Z using VCM as raw material. In this Company Z, during the 3-year period under study, there were 6 documented cases of hepatitis and 5 cases of primary liver cancer.

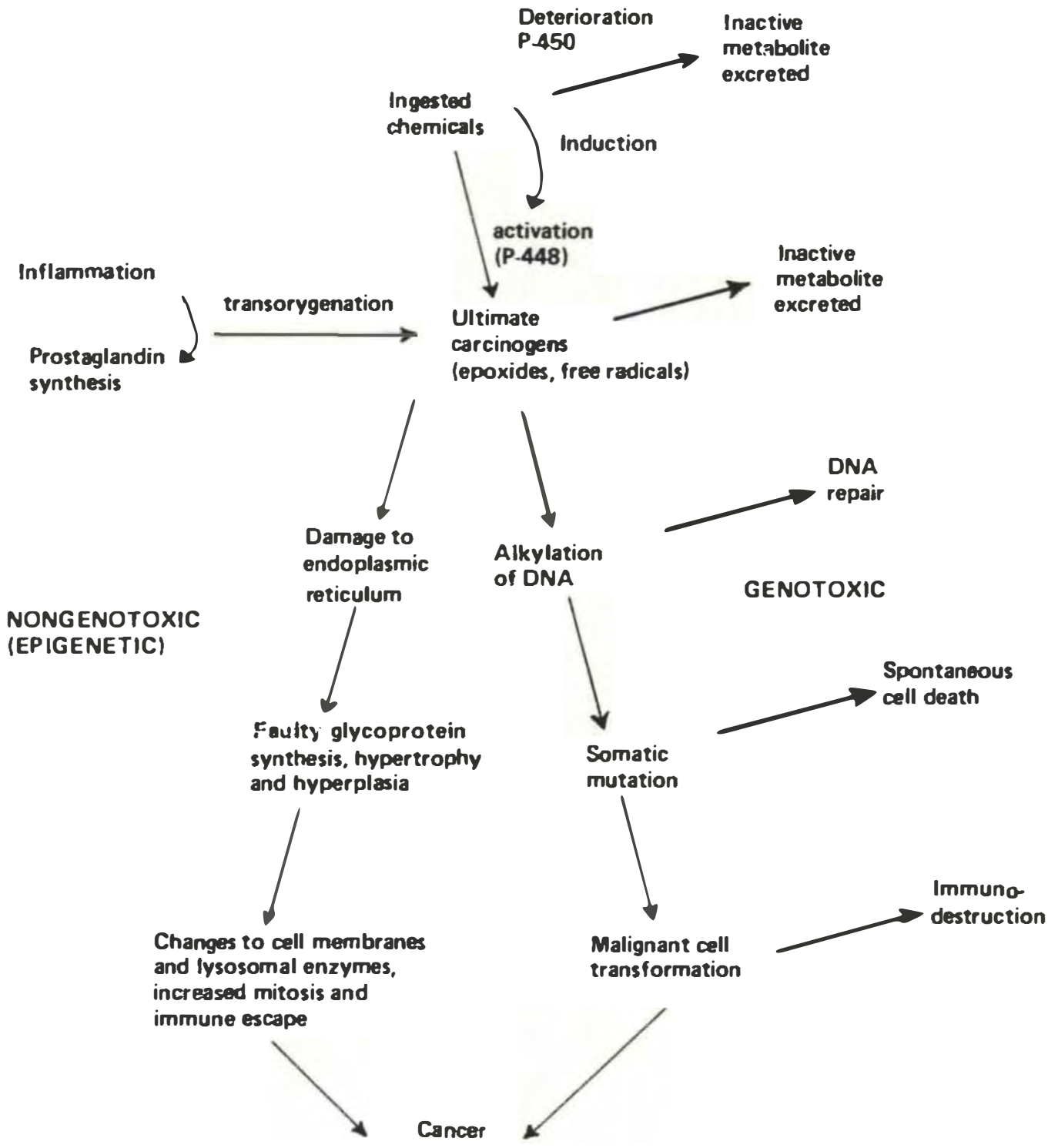
Going by the best estimates of the incidence of Hepatitis B and Hepatocellular Carcinoma coming from the Comprehensive Study of Primary Carcinoma of the Liver and other related Liver Diseases in the Philippines (Domingo, E. *et al.*, 1985) and other published estimates, the incidence of Hepatitis and Hepatocellular Carcinoma that we found in Company Z is way in excess of expectation as shown in Table 11.

At this point, in order to appreciate better the complexity of risk assessment in human cancer, I would like to present the current state of knowledge of the molecular mechanisms of chemical carcinogenesis as summarized by Dr. D.V. Parke in 1983. (Fig. 1 – Chemical carcinogenesis)

If we start at the point when the ultimate carcinogens impinges on the organism, we can see that *free radicals and active oxygen* as produced for instance by exposure to ionizing radiation are active initiating factors as are *chemical reactive intermediates,* such as *epoxides* of Benzo pyrene which can covalently bind to DNA and thus result in somatic mutations. According to recent theory, one hit at DNA is insufficient, and a major damage to DNA involving substantial transposition of genetic material and multiple invitations are required before malignant cell transformation can occur.

As shown at the top of the diagram, ingested potential chemical carcinogen may be *detoxified and inactivated* through the usual P-450 dependent microsomal mixed-function oxidase, *or activated* by Cytochrome P-448, if its stereochemistry allows sterically hindered oxygenation. For instance, pre-treatment of animals with phenobarbital leads to induction of cytochrome P-450 and the inactivation of the potential carcinogen benzopyrene to the ultimate carcinogen the *reactive epoxide form.* The sequence of events that lead to cancer may be through genetic damage (GENOTOXIC) or the so-called initiation phase, or through the promotion stage – which is often associated with non-genotoxic or epigenetic mechanisms, or with enzyme-inducing chemicals which promote the propagation of any malignant cell transformation through induction of hypertrophy and hyperplasia. The non-genotoxic mechanisms involve interaction and damage to subcellular structures such

Fig. 1. Molecular mechanism of chemical carcinogenesis*



*By D. V. Parke

Table 11. Incidence of hepatitis and hepatocellular carcinoma in Company Z compared with estimates of incidence rates published in the literature

Condition	*Incidence Rate*	*Investigators*	*Expected Number for 419 employees of Company Z*	*Adj. 3 yr.*		
				Actual	*Exp.*	*Act./Exp.*
Hepatitis B	70/100,000/yr.	Domingo *et al.*, 1985	0.29/yr.	6	.87	6.9x
Hepatocellular Carcinoma	40/100,000/yr.	Domingo *et al.*, 1985	0.17/yr.	5	.51	9.8x
Primary Liver Cancer Among Males	20/100,000/yr.	Bulatao-Jayme *et al.*. 1982	0.084/yr.	5	.25	20x

as endoplasmic reticulum mitochondria and cysosomes. Thus, malignant cell transformation results from an extensive generalized damage of the cell and not just from the alkylation of DNA.

Various chemicals and factors may enhance or diminish carcinogenesis by affecting any or a combination of the molecular mechanisms as shown by the processes to the right of the dark arrows on the figure.

Keeping this figure in mind, we can now examine some of the etiologic factors which may be operative in the primary liver cancer among workers in the plastic industry in Philippine setting.

1) *VCM exposure*

It is well accepted that chronic exposure to high levels of VCM, say at 5-10 ppm for 10-15 years, is associated with increased risk to liver disorders, including primary liver cancer. The occurrence of a specific type of malignancy, namely Angiosarcoma of the Liver, is considered almost pathognomonic of VCM exposure. The spot sampling of air for VCM showed that the Threshold Value Limit was exceeded in some places in this Company.

From this study, the only documented pathologically diagnosed care of Angiosarcoma of the Liver, slides of which I had presented earlier, was in a mason worker from Pardo, Cebu. Although he worked with plastic compounds as a mason, he was never an employee in a VCM-using plastic Company. The other case of Angiosarcoma of the Liver was included in the Central Tumor Registry of Metro Manila Hospitals, but we could not trace the medical records of the case to get further specific information on the case.

The molecular mechanisms of carcinogenesis by vinyl chloride is still unknown but it can be both at the genotoxic or non-genotoxic stages.

2) *Hepatitis B*

According to Dr. C.W. Chan of Hongkong, the majority of hepatocellular carcinomas in Southeast Asian countries are now considered causally related to the prevalence of Hepatitis B virus in the region. The comprehensive Liver Study estimates the prevalence of Hepatitis B in Filipinos at 8.7% of the population, one of the highest in the world. The 6 cases of Hepatitis over 3 years in Company Z is 7x more than the expected number in the general population.

The molecular mechanism of hepatocarcinogenesis by Hepatitis B Virus infection is still unknown but liver hepatoma is one of the cancers now considered to be etiologically associated with a virus infection.

3) *Aflatoxin*

A series of reports from Bulatao-Jayme, J. *et al.* (1978-1982) has established the probable contribution of dietary load of the mycotoxin-aflatoxin in the increased risk of developing primary liver cancer. Using the odds ratio as an estimate of relative risk, she has estimated that the very heavy aflatoxin dietary load of 7 mcg or higher per day increased the risk of developing Primary Liver Cancer 17 times as shown in Table 12. (Bulatao-Jayme, *et al.* 1982)

Table 12. Dose-response relationship in terms of relative risk of developing PLC by category of overall mean aflatoxin load

Category of Overall Mean Aflatoxin Load	*Number of subjects*		*Relative risk*[b] *(Odds Ratio)*
	Cases (90)[a]	*Controls (90)*[a]	
Light (0 – 3 mcg)	20	74	1.0
Mod. Heavy (4 – 6 mcg)	15	4	13.9*
Very Heavy (7 mcg & over)	55	12	17.0*

( ) a = number of subjects
* = significant
b = Relative Risk/Odds Ratio $= \frac{\text{Cases}_H \times \text{Controls}_L}{\text{Cases}_L \times \text{Controls}_H}$

Together with nitrosoamines, aflatoxin constitute the two major carcinogens that contaminate human foodstuff particularly in the tropical regions of the world. Improvement in agricultural and storage practices can greatly reduce the levels of contamination from levels measured in parts per million to lower levels measured in parts per billion with a commensurate reduction in cancer risk.

Our export of copra meal to Europe is now being threatened because our aflatoxin levels in copra may not meet the stricter standards being contemplated by the European community.

4) *Alcohol*

Bulatao-Jayme in the same paper cited above, found that the combination of aflatoxin and alcohol intake produced a strong synergistic carcinogenic effect to as high as 35x with Heavy Aflatoxin and Heavy Alcohol Intake compared to Light Aflatoxin and Light Alcohol Intake as shown in Table 13.

Table 13. Relative risk of developing PLC from the combined effects of aflatoxin load and alcohol intake

Category of aflatoxin load and alcohol intake	*Relative risk*
Light Aflatoxin, Light Alcohol	1.0
Light Aflatoxin, Heavy Alcohol	3.9*
Heavy Aflatoxin, Light Alcohol	17.5*
Heavy Aflatoxin, Heavy Alcohol	35.0*

* = significant

Alcoholic beverages contain nitrosamines mycotoxins and other known carcinogens in significant amounts. Additionally alcohol has been shown to inhibit prosta-glandin activity and mucus production in the GI Tract and may thus promote carcinogenesis.

Other factors like cigarette smoking and nutrition may also play significant roles in the development of liver cancer in the Philippine setting.

Nicotine and other components of tobacco smoke resulting in the depletion of intracellular glutathione resulting in the opening of tight junctions of epithelial cells, thus carcinogens may penetrate to the sensitive basal cell layer.

Malnutrition and protein deficiency results in inadequate synthesis of detoxifying enzyme systems and cellular defense mechanisms dependent on glutathione.

With all four etiologic factors apparently operative, how much can one attribute to any or a combination of any of these factors. Are the factors when present in a given case simply additive or do they interact in a synergistic way.

Given the complexity of human disease and the multiplicity of factors that determine the tendency or risk of getting it ranging from the genetic or constitutional factor to the myriad environmental influences, risk assessment of human disease conditions will always remain problematic.

Although risk analysis and risk benefit analysis using probabilistic Risk Assessment have been proven useful in many diverse fields of application such as nuclear power plant accidents, dam operation accidents (Elizabeth-Pate, 1983) or even in land use planning (Popper 1983) its use in multi-factorial risk assessment such as in Primary Liver Cancer appears a long way off, if at all it is possible.

Acknowledgment

This research project is supported through a financial grant from the Philippine Council for Health Research and Development.

The cooperation of the following institutions and associations are hereby acknowledged:

1. National Institute of Science and Technology, NSTA
2. Ministry of Labor and Employment, particularly, the Bureau of Working Conditions
3. Ministry of Health
4. University of the Philippines, Institute of Public Health
5. Philippine Occupational and Industrial Medical Association
6. Philipine Cancer Society
7. The Companies of the Plastic Industry included in this study

The other personnel of this project are:

1.	Dr. Eulalia L. Venzon	—	Assistant Project Leader; Co-researcher
2.	Dr. Francisco R. Jose	—	Consultant
3.	Lucila C. Sumalinog	—	Research Assistant
4.	Yolanda C. Paras	—	Research Assistant
5.	Alexandra J. Fojas	—	Research Assistant
6.	Ingrid R. Llave	—	Research Assistant

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